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Exposure and Risk Assessment for Benzo(a)pyrene and Other Polycyclic Aromatic Hydrocarbons. Volume 1. Summary

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Water

An Exposure and Risk Assessment for Benzo[a]pyrene and Other Polycyclic Aromatic Hydrocarbons

Volume I. Summary

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16. Abstract (Limit: 200 words)

This report assesses the risk of exposure to polycyclic aromatic hydrocarbons (PAHs). This is Volume I of a four-volume report, summarizing an analysis of 16 PAHs: benzo[a]pyrene, naphthalene, anthracene, acenaphthene, fluoranthene, fluorene, phenanthrene, pyrene, acenaphthylene, benz[a]anthracene, benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[g,h,i]perylene, chrysene, dibenz[a,h]anthracene, and indeno[1,2,3-c,d]pyrene. This study is part of a program to identify the sources of and evaluate exposure to 129 priority pollutants. The analysis is based on available information from government, industry, and technical publications assembled in July of 1982.

The assessment includes an identification of releases to the environment during production, use, or disposal of the substances. In addition, the fate of PAHs in the environment is considered; ambient levels to which various populations of humans and aquatic life are exposed are reported. Exposure levels are estimated and available data on toxicity are presented and interpreted. Information concerning all of these topics is combined in an assessment of the risks of exposure to PAHs for various subpopulations.

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Exposure
Risk
Water Pollution
Air Pollution
Effluents
Waste Disposal
Food Contamination
Toxic Diseases
Polycyclic Aromatic Hydrocarbons
Benz[a]anthracene
Benzo[b]fluoranthene
Benzo[k]fluoranthene
Benzo[g,h,i]perylene
Dibenz[a,h]anthracene
Indeno[1,2,3-c,d]pyrene
Phenanthrene
Acenaphthylene
Pyrene
Benzo[a]pyrene
Naphthalene
Anthracene
Acenaphthene
Fluoranthene
Fluorene
Chrysene
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AN EXPOSURE AND RISK ASSESSMENT FOR BENZO[a]PYRENE AND OTHER POLYCYCLIC AROMATIC HYDROCARBONS: VOLUME I. SUMMARY

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OFFICE OF WATER REGULATIONS AND STANDARDS
OFFICE OF WATER
U.S. ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

FOREWORD

Effective regulatory action for toxic chemicals requires an understanding of the human and environmental risks associated with the manufacture, use, and disposal of the chemical. Assessment of risk requires a scientific judgment about the probability of harm to the environment resulting from known or potential environmental concentrations. The risk assessment process integrates health effects data (e.g., carcinogenicity, teratogenicity) with information on exposure. The components of exposure include an evaluation of the sources of the chemical, exposure pathways, ambient levels, and an identification of exposed populations including humans and aquatic life.

This assessment was performed as part of a program to determine the environmental risks associated with current use and disposal patterns for 65 chemicals and classes of chemicals (expanded to 129 "priority pollutants") named in the 1977 Clean Water Act. It includes an assessment of risk for humans and aquatic life and is intended to serve as a technical basis for developing the most appropriate and effective strategy for mitigating these risks.

This document is a contractors' final report. It has been extensively reviewed by the individual contractors and by the EPA at several stages of completion. Each chapter of the draft was reviewed by members of the authoring contractor's senior technical staff (e.g., toxicologists, environmental scientists) who had not previously been directly involved in the work. These individuals were selected by management to be the technical peers of the chapter authors. The chapters were comprehensively checked for uniformity in quality and content by the contractor's editorial team, which also was responsible for the production of the final report. The contractor's senior project management subsequently reviewed the final report in its entirety.

At EPA a senior staff member was responsible for guiding the contractors, reviewing the manuscripts, and soliciting comments, where appropriate, from related programs within EPA (e.g., Office of Toxic Substances, Research and Development, Air Programs, Solid and Hazardous Waste, etc.). A complete draft was summarized by the assigned EPA staff member and reviewed for technical and policy implications with the Office Director (formerly the Deputy Assistant Administrator) of Water Regulations and Standards. Subsequent revisions were included in the final report.

Michael W. Slimak, Chief
Exposure Assessment Section
Monitoring & Data Support Division (WH-553)
Office of Water Regulations and Standards

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1.0 INTRODUCTION

The Office of Water Regulations and Standards (OWRS), Monitoring and Data Support Division, of the U.S. Environmental Protection Agency is conducting a program to evaluate the exposure to and risk of 129 priority pollutants in the nation's environment. The risks to be evaluated include potential harm to human beings and deleterious effects on fish and other biota. The goals of the program under which this report has been prepared are to integrate information on cultural and environmental flows of specific priority pollutants, to estimate the likelihood of receptor exposure to these substances, and to evaluate the risk resulting from such exposures. The results are intended to serve as a basis for estimating the magnitude of the potential risk and developing a suitable regulatory strategy for reducing any such risk.

This report, comprised of four separate volumes, provides a summary of the available information concerning the releases, fate, distribution, effects, exposure, and potential risks of the 16 priority pollutants that are polycyclic aromatic hydrocarbons (PAHs). The chemical structures of these compounds are shown in Figure 1-1.

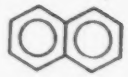
The number of chemicals considered in this exposure and risk assessment is appreciable. The possibility of preparing 16 separate exposure and risk assessment documents was considered and rejected because it would lead to considerable redundancy and because so little information was available on some of the individual PAHs. As an alternative, the 16 PAHs were organized at the onset of the work into three groups, as indicated in Figure 1-1.

The rationale for the organization into these three specific groups included considerations of materials balance, chemical properties related to fate and environmental pathways, and health effects, as described briefly below.

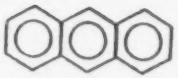
- Naphthalene is the only one of the 16 PAHs with substantial U.S. commercial production and with a significant potential for direct exposure to consumers of a commercial product (mothballs). It is significantly more volatile and more water soluble than any other PAH. It was not anticipated to have carcinogenic effects in humans.

- Anthracene, acenaphthene, fluorene, fluoranthene, phenanthrene and pyrene are all imported in rather small quantities for special commercial uses. These compounds are three- and four-ring PAHs, with moderately low volatility and water solubility. The question of their possible carcinogenicity was expected to require careful review. Most of the information pertaining to this group is specific to anthracene.

NAPHTHALENE



THE ANTHRACENE GROUP



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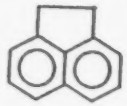

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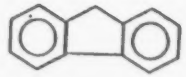

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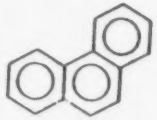
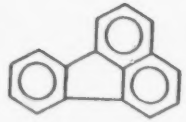


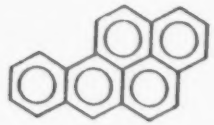
Phenanthrene

Pyrene

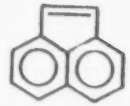


Fluoranthene

THE BENZO[a] PYRENE GROUP



Benzo[a] pyrene



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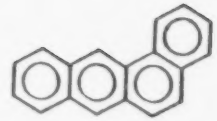
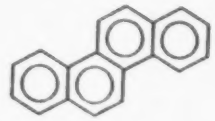


Benz[a] anthracene



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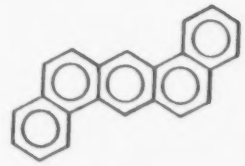
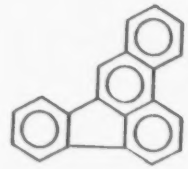
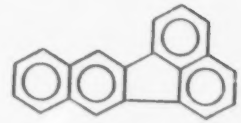


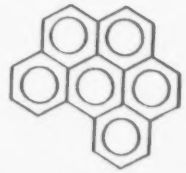
Dibenz[a,h] anthracene



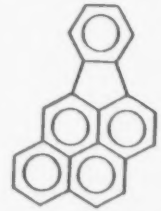
Benzo[b] fluoranthene



Benzo[k] fluoranthene



Benzo[g,h,i] perylene



Indeno[1,2,3-c,d] pyrene

FIGURE 1-1 STRUCTURES OF THE PRIORITY POLLUTANT POLYCYCLIC AROMATIC HYDROCARBONS

- Benzo[a]pyrene (BaP) and the eight other PAHs in the third group have no commercial production or use, except as research laboratory standards. They are released to the environment inadvertently by combustion sources. With one exception (acenaphthylene), the chemicals in this group have very low vapor pressures and water solubilities. Several of the PAHs in the BaP group had been identified as carcinogens. Much of the information regarding this group of compounds is for BaP.

The exposure and risk assessment for each of the three groups of PAHs was treated in a separate chapter of a multivolume report; Chapter 3.0 (Volume II) concerns naphthalene; Chapter 4.0 (Volume III) concerns the anthracene group PAHs; and Chapter 5.0 (Volume IV) concerns the benzo[a]pyrene group PAHs. These chapters are bound separately.

Potential waterborne routes of exposure are the primary focus of these exposure and risk assessments because of the emphasis of OWRS on aquatic and water-related pathways. Inhalation exposures are also considered, however, in order to place the water-related exposures into perspective. Each chapter contains major sections discussing the following topics:

- Information on environmental releases of the subject PAHs, including the form and amounts released and the receiving medium at the point of entry into the environment (materials balance);

- Description of the fate processes that transform and/or transport the compounds from the point of release through environmental media until exposure of humans and other receptors occurs, and a summary of reported concentrations detected in the environment, with a particular emphasis on aquatic media;

- Discussion of the available data concerning adverse health effects of the subject PAHs on humans, including (where known) the doses eliciting those effects and an assessment of the likely pathways and levels of human exposure;

- Review of available data concerning adverse effects on aquatic biota and the levels of environmental exposure; and

- Discussion of risk considerations for various subpopulations of humans and other biota.

Two comments regarding the materials balance section are appropriate. First, these sections were based in large part on draft material prepared by Acurex Corporation, under EPA Contract 68-01-6017, and provided to Arthur D. Little, Inc. by EPA. Second, the phrase "materials balance" is somewhat inappropriate when applied to chemicals such as the PAHs that are produced primarily as byproducts of combustion

processes. Since most PAH production is inadvertent rather than deliberate commercial production, the conventional approach of trying to balance production versus use and environmental release is not strictly applicable to these chemicals. Therefore, the materials balance sections of these exposure and risk assessments are focused on estimates of releases from major sources such as combustion; considerable uncertainty is associated with most of these estimates.

After an initial review of the three exposure and risk assessments covering all 16 priority pollutant PAHs, it was determined that one chemical, benzo[a]pyrene, was of appreciably greater interest to OWRS than were the other 15 compounds studied. This interest reflects the more extensive data base available for assessment of environmental fate and exposure and also the existence of some, although limited, dose-response data to which various extrapolation models can be applied for estimation of potential human carcinogenic risk from ingestion of BaP. For the other PAHs considered, data on carcinogenic or other long-term effects were generally limited, nonquantitative, and/or did not indicate statistically positive results. Table 1-1 presents a summary of the hazard of the 16 priority pollutant PAHs in terms of carcinogenicity, based on qualitative review of available information.

For these reasons, the technical summary presented in this volume (Volume I) is organized somewhat differently than are Chapters 3.0-5.0 (Volumes II-IV). The summary is focused on benzo[a]pyrene as the PAH of greatest interest. The estimated releases to the environment, environmental fate, monitoring data, human effects and exposure, biotic effects and exposure, and risk considerations concerning BaP are presented in expanded summary form. Abbreviated summaries are then provided for naphthalene, anthracene group PAHs, and the other PAHs considered.

Critical data and selected references to the literature are included in this summary volume so that it may be read and understood without reference to the separately bound Chapters 3.0-5-0. The latter volumes contain more extensive compilations of data, more detailed discussions of the available information and of the interpretations drawn, and more complete documentation of the multiple literature sources that were reviewed in the course of this work.

TABLE 1-1. SUMMARY OF EVIDENCE FOR CARCINOGENICITY OF PRIORITY POLLUTANT PAHs

PAH*	Basis
Benzo[a]pyrene	Positive oral carcinogen with other positive carcinogenic data.
Dibenz[a,h]anthracene	Positive oral carcinogen with other positive carcinogenic data.
Benz[a]anthracene	Positive oral carcinogen with other positive carcinogenic data.
Benzo[g,h,i]perylene	Not tested orally, other positive carcinogenic or co-carcinogenic data.
Benzo[b]fluoranthene	Not tested orally, other positive carcinogenic or co-carcinogenic data.
Chrysene	Not tested orally, other positive carcinogenic or co-carcinogenic data.
Indeno[1,2,3-c,d]pyrene	Co-carcinogen or initiator with negative carcinogen or *in vivo* mutagen.
Pyrene	Co-carcinogen or initiator with negative carcinogen or *in vivo* mutagen.
Fluoranthene	Co-carcinogen or initiator with negative carcinogen or *in vivo* mutagen.
Benzo[k]fluoranthene	Negative in a single carcinogenic study.
Phenanthrene	Several negative carcinogenic and mutagenic studies but not tested orally.

TABLE 1-1. SUMMARY OF EVIDENCE FOR CARCINOGENICTY OF PRIORITY POLLUTANT PAHs (Continued)

Anthracene	Negative studies, tested orally.
Naphthalene	Negative studies, tested orally.

*No data for evaluation of carcinogenicity were available for acenaphthene, acenaphthylene, or fluorene.

2.0 TECHNICAL SUMMARY

2.1 BENZO[a]PYRENE (BaP)

2.1.1 Risk Conclusions

BaP is suspected of being a human carcinogen, although there are a number of limitations regarding the experimental animal data upon which this conclusion is based. Table 2-1 shows estimates of the relative potential carcinogenic risks (excess lifetime tumor probability) for the major BaP exposure routes determined by using a range of risk calculated using two sets of animal data and three dose-response extrapolation models. Although these two sets of animal data demonstrate a carcinogenic response to BaP in mice by the ingestion route, the quantitative estimates of human risk must be treated with considerable caution since they are dependent on a number of assumptions that seriously limit the degree of confidence associated with the quantitative human risk extrapolations for BaP. Moreover, there is, as always, no clear agreement over the most appropriate model for performing such extrapolations. Additional uncertainty is introduced into the risk estimates by the conversion techniques used to estimate human equivalent doses, and by possible differences in susceptibility between humans and mice. Due to the use of a number of conservative assumptions in the risk calculations, the results shown in Table 2-1 most likely over-estimate the actual risk to humans.

The highest potential carcinogenic risks appear to be associated with cigarette smoking. For non-smokers, the highest potential carcinogenic risks are associated with dietary exposure to BaP (notably ingestion of charcoal-broiled meats and fish). Inhalation exposures at the upper end of the range in urban areas are higher than dietary exposure, but the estimates are based upon limited monitoring data. Drinking water generally represents a less significant source of BaP exposure than the diet or inhalation.

BaP has also been shown to induce *in vivo* chromosomal aberrations in laboratory animals. Because of this indication of mutagenicity, BaP exposure could be expected to contribute to the genetic burden of a population; however, since extrapolation procedures for genetic risks have not been well established, a quantitative risk assessment for these kinds of adverse health effects is not presently feasible. Little information is available on other health effects of BaP.

No conclusions could be drawn as to the risk to biota associated with BaP exposure due to the lack of information regarding effects. Similarly, no water quality criterion for aquatic life has been set for this compound.

TABLE 2-1. ESTIMATED RANGES OF CARCINOGENIC RISK TO HUMANS DUE TO BENZO[a]PYRENE EXPOSURE FOR VARIOUS ROUTES

Route	Average Lifetime BaP Exposure (μg/day)	Size of Exposed Population	Estimated Lifetime Excess Probability of Cancer[a]	Estimated Incidence (excess cancers/year)
Typical Diet	0.05	221×10^6	4×10^{-6} to 6×10^{-4}	13 - 1,900
Drinking Water	0.0006	221×10^6	1×10^{-10} to 7×10^{-6}	<< 1 - 22
Ambient Air - Urban	0.02 - 2	166×10^6	6×10^{-7} to 4×10^{-2}	1 - 95,000
- Rural	0.0002 - 0.2	55×10^6	3×10^{-11} to 3×10^{-3}	<< 1 - 2,400
Smoking	0.6[b]	54×10^6[b]	3×10^{-4} to 1×10^{-2}	230 - 7,700

[a] A range of probability is given, based on several different dose-response extrapolation models. The lifetime excess probability of cancer represents the increase in probability of cancer over the normal background incidence, assuming that an individual is continuously exposed to BaP at the indicated daily intake over a 70-year lifetime. There is considerable variation in the estimated risk due to uncertainty introduced by the use of laboratory rodent data, by the conversion to equivalent human dosage, and by the application of hypothetical dose-response curves. In view of several conservative assumptions that were utilized, it is likely that these predictions overestimate the actual risk to humans.

[b] It was assumed that the total population of smokers (54 million) smoked on average 25 cigarettes per day. From 25% to 30% of smokers consume more than 25 cigarettes per day and consequently may receive a higher daily exposure.

Source: Data taken from Volume IV of this report.

2.1.2 Releases of BaP to the Environment

Combustion is the major source of environmental loading of BaP. Residential heating is the single largest combustion source, since these emissions are largely uncontrolled. Table 2-2 summarizes the estimates of BaP releases from these sources and the emission factors that were used in the calculation.[1]

BaP is also released to the environment as a result of its presence in petroleum- and coal-derived oils, fuels, and solvents. Coal tar production and distillation released an estimated total of about 4 kkg BaP in 1978.[2] Creosote oil is used in the wood preserving industry; however, less than 1 kkg BaP has been estimated to be released from this source in 1978 (U.S. EPA 1979a).

Publicly-Owned Treatment Works (POTWs) also represent a source of BaP releases to the environment. The volume of releases is largely dependent upon variations in industrial discharges to POTWs. On the basis of a total POTW flow of approximately 10^{11} l/day (U.S. EPA 1978a) and average BaP concentrations of 0.1 µg/l in the influent (it was not detected in the effluent), the environmental releases can be estimated. The total environmental loading of BaP from this source was calculated to be 3.7 kkg/yr. The amount contained in sludge was calculated to be less than 1 kkg, assuming 6.0×10^{5} kkg dry sludge produced annually (U.S. EPA 1976 and a concentration of 3.9 µg/kg in the sludge (U.S. EPA 1980a). The difference between the total environmental loading and the BaP in the sludge was allocated as an atmospheric release. However, an unknown fraction of this 3.7 kkg may in fact be present in the POTW effluent at concentrations too low to be detected.

Table 2-3 summarizes the releases of BaP to the environment including those from combustion and other sources. Considerable uncertainty is associated with all of these release estimates. BaP is largely a combustion product or present at low levels in coal tar and petroleum. Since this compound is not directly produced, the base of information on which to apply emission factors does not exist. There are very limited monitoring data, and the representativeness of the emission factors is unknown. Therefore, these estimates should be viewed as an indication of the relative magnitude of BaP releases to the environment compared with other compounds, and for identifying roughly the sources that are important.

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[1]These estimates were developed by Acurex, Inc. for EPA under Contract No. 68-01-6017. Their data are summarized in Volume IV of this report.

[2]See footnote 1.

TABLE 2-2. ESTIMATED ANNUAL AIR EMISSIONS OF BENZO[a]PYRENE FROM COMBUSTION SOURCES, 1978

Source	Emission Factor	Amount Combusted	Estimated Releases (kkg)
Residential Coal Combustion	0.0015 g/kg	8.9×10^6 kkg	10
Fireplaces	0.0008 g/kg	2.9×10^6 kkg	2
Primary Residential Wood Heating	0.004 g/kg	6.9×10^6 kkg	30
Auxiliary Residential Wood Heating	0.004 g/kg	9.2×10^6 kkg	40
Prescribed Burning	0.740 mg/kg (dry fuel)	36×10^6 kkg (dry wt.)	30
Wildfire	0.740 mg/kg (dry fuel)	2×10^7 kkg (dry wt.)	20
Agricultural Burning	0.740 mg/kg (dry fuel)	13×10^6 kkg (dry wt.)	10
Gasoline	8 ug/mile	1.7×10^{12} miles traveled, 67% Emission reduction	10
Tire Wear	[a]	[a]	1
Utility Boilers - Coal	[b]	4.8×10^8 kkg	1
Oil	[b]	7.8×10^7 kkg	1
Incinerators	0.04 ug/kg refuse	municipal - 385 kkg/day	<1
		commerical - 8×10^6 kkg/yr	<1
Coal Refuse Pile	0.005 kg/kg POM	[c]	<1
Cigarettes	0.01 ug/cigarette	6.2×10^{11} cigarettes	<1
			Total 159

[a] 1.7×10^{12} miles traveled, 0.19 g/vehicle mile airborne particulates, 0.15 g/vehicle-mile is deposited on road surface, rubber - 33% carbon black, 17 ug BaP/g carbon black.

[b] Described in Volume IV of this Report.

[c] 190×10^6 m^3 refuse pile volume (21% burning), density 1.5 kkg/m^3 POM emission rate of 1.3×10^{-8} kg/kkg-hr.

Source: Acurex Materials Balance - Draft Report prepared under Contract No. 68-01-6017. Data summarized in Volume IV of this report.

TABLE 2-3. SOURCES OF BENZO[a]PYRENE TO THE ENVIRONMENT, 1978

Source	Release (kkg)			
	Air	Water	POTW	Land
Combustion	160	-	-	-
Coal tar production	2	0.7	0.9	0.6
POTW	4	not detected	-	<1
TOTAL	166	0.7	0.9	<2

Source: Acurex. Materials Balance - Draft Report Prepared under Contract No. 68-01-6017. Data Summarized in Volume IV of this Report.

2.1.3 Fate and Distribution of BaP in the Environment

2.1.3.1 Transport to the Aquatic Environment

Benzo[a]pyrene is released to the environment primarily as a product of combustion as discussed above. Since virtually all of the BaP in the atmosphere is adsorbed onto particulates, deposition on land and water is likely to be an important fate process. The air-to-surface transport pathway was examined using the method developed by Arthur D. Little, Inc. (1981). The results, shown in Table 2-4, indicate that an estimated 25% of the BaP air emissions will be deposited annually on the surface of the U.S., or about 40 kkg. If approximately 2% of the total area of the U.S. is surface water, less than 1 kkg BaP annually would be directly deposited on inland surface waters from the atmosphere. An additional amount would eventually reach surface waters via surface runoff, although the total is estimated to be less than 1 kkg annually. The effect on water bodies near combustion sources may be more significant. The BaP remaining in the atmosphere is likely to be rapidly photolyzed. However, a thorough examination of the atmospheric fate of these compounds was beyond the scope of this report.

2.1.3.2 Fate in the Aquatic Environment

The basic physicochemical properties of BaP are shown in Table 2-5. The compound's vapor pressure and Henry's law constant suggest that volatilization from surface waters will not be an important pathway for this compound. In fact, a volatilization half-life of 430 hours has been measured in a simulated aquatic environment (Southworth 1979). Calculated half-lives (using a one-compartment model) ranged from 140 hours to 700 hours, depending upon the type of water body (Smith et al. 1978).

Partition coefficient and solubility data suggest that BaP in the aquatic environment is likely to be adsorbed onto sediment and biota, and this is probably a predominant removal mechanism from the water column. Concentrations predicted from a one-compartment model show that concentrations of BaP on sediments and suspended solids are at least ten times greater than dissolved concentrations (Smith et al. 1978).

The BaP remaining in the water column may be oxidized. Several authors (Andelman and Suess 1970, Smith et al. 1978, NAS 1972, Neff 1979) have suggested that direct photo-oxidation of BaP is a major oxidation pathway in water. A calculated half-life of 1.2 hours for midday photolysis in winter is in close agreement with the measured results of 1.1 hours and 0.7 hours, although the measured half-lives were found to be somewhat longer in the presence of humic acid (Smith et al. 1978).

BaP is concentrated in aquatic organisms, with bioconcentration factors up to about 140,000 reported (Lu et al. 1977). Although significant concentrations have been reported in aquatic organisms,

TABLE 2-4. EVALUATION OF AIR-TO-SURFACE PATHWAY FOR BENZO[a]PYRENE

	Rural	Urban	Near Combustion Source
Fraction of airborne mass adsorbed on particulate	0.99	1	
Dry deposition velocity (cm/sec)	1	1	
Precipitation scavenging ratio: $\frac{ng/l\ (water)}{\mu g/m^3\ (air)}$	$6x10^4$	$6x10^4$	$1.2x10^5$[a]
Percent of atmospheric emissions deposited			
- dry deposition	22	19	
- wet deposition	4	4-7	
- total	26	23-26	

[a]Depends on the proximity to a combustion source.

Source: Data taken from Volume IV of this report.

TABLE 2-5. BASIC PHYSICOCHEMICAL PROPERTIES OF BENZO[a]PYRENE

Formula	$C_{20}H_{12}$
Molecular Weight	252.32
Melting Point	179°C
Vapor Pressure	5.6×10^{-9} torr at 25°C
Water Solubility	0.0038 mg/l at 25°C
Log octanol/water Partition Coefficient	6.08 at 25°C
Henry's Law Constant	4.89×10^{-7} atm m^3/mole at 25°C

Source: SRI (1980); Versar, Inc. (1979).

bioaccumulation is not expected to be a significant loss mechanism from water. In addition, microbial degradation does not appear to be an important fate process for BaP (Gardner et al. 1979, Lu et al. 1977, Schwall and Herbes 1978).

2.1.3.3 Modeling of Environmental Distribution

The Mackay equilibrium partitioning model (Mackay 1979) was used to estimate the partitioning of BaP in the environment. This application is described in Volume IV of this report. The results suggest that 99.9% of the BaP introduced into the air-water sediment system will reside in the sediment compartment. Less than 0.1% will be present in the water column, and less than 0.001% will be partitioned to the atmosphere.

The U.S. EPA EXAMS model (U.S. EPA 1980b) was also used to estimate BaP fate in aquatic systems using a loading rate of 0.1 kg/hour. Table 2-6 summarizes the fate of BaP in generalized aquatic systems modeled by EXAMS. The ultimate fate of BaP in these systems is controlled primarily by chemical processes in the static systems, and physical transport beyond the system boundaries for the river systems. The persistence of BaP in aquatic systems is reflected in the time for self-purification predicted by EXAMS. The necessary time for removal of 97% of the BaP accumulated in the turbid river system was estimated as 89 days, while the analogous self-purification time predicted for the static pond system was as high as 69 years.

Figure 2-1 summarizes the major inputs of BaP to the aquatic environment, as well as the dominant fate and transport pathways.

2.1.3.4 Concentrations of BaP in the Environment

BaP has been monitored in environmental media. Most of the observations reported in STORET (the U.S. EPA water quality data base) are below the detection limits, which are generally 10 µg/l. Data from other sources indicate that BaP levels in waters are generally less than 1 µg/l and commonly less than 0.01 µg/l (White and Vanderslice 1980).

Reported levels of BaP in sediment are much higher than in water. In the STORET data base, levels up to 1,400 µg/kg have been recorded, although only 11 of 125 observations were above the detection limit (often greater than 1,000 µg/kg) (U.S. EPA 1980c). Hites et al. (1977) reported a high level of 8,000 µg/kg BaP (dry weight) in sediment from the Charles River in Massachusetts. Concentrations in urban soils appear to be about two orders of magnitude higher than concentrations in rural soil (White and Vanderslice 1980).

Concentrations of BaP in drinking water are reported to range from 0.2 ng/l to 1.6 ng/l, with the highest reported BaP concentration found in drinking water from New Orleans, LA (Basu and Saxena 1978).

TABLE 2-6. FATE OF BENZO[a]PYRENE IN GENERALIZED AQUATIC SYSTEMS[a]

System	Percent Distribution		Percent Lost by Various Processes				
	Residing in Water at Steady-State	Residing in Sediment at Steady-State	Transformed by Chemical Process	Transformed by Biological Process	Volatilized	Lost by Other Processes[b]	Time for System Self-Purification[c]
Pond	0.24	99.76	68.79	0	0.16	31.03	69 years
Eutrophic Lake	1.85	98.15	95.10	0.05	0.21	4.64	25 years
Oligotrophic Lake	18.75	81.25	99.97	0	0.02	0.01	393 days
River	2.17	97.83	0.04	0	0	99.96[b]	95 days
Turbid River	5.63	94.37	0	0	0	100[b]	89 days
Coastal River	0.56	99.44	2.89	0	0.02	97.08[b]	376 days

[a]All data simulated by the EXAMS model (U.S. EPA 1980b).

[b]Primarily loss through physical transport beyond system boundaries.

[c]Estimate for removal of ca. 97% of the toxicant accumulated in system. Estimated from the results of the half-lives for the toxicant in bottom sediment and water columns, with overall cleansing time weighted according to the pollutant's initial distribution.

Source: Data taken from Volume IV of this report.

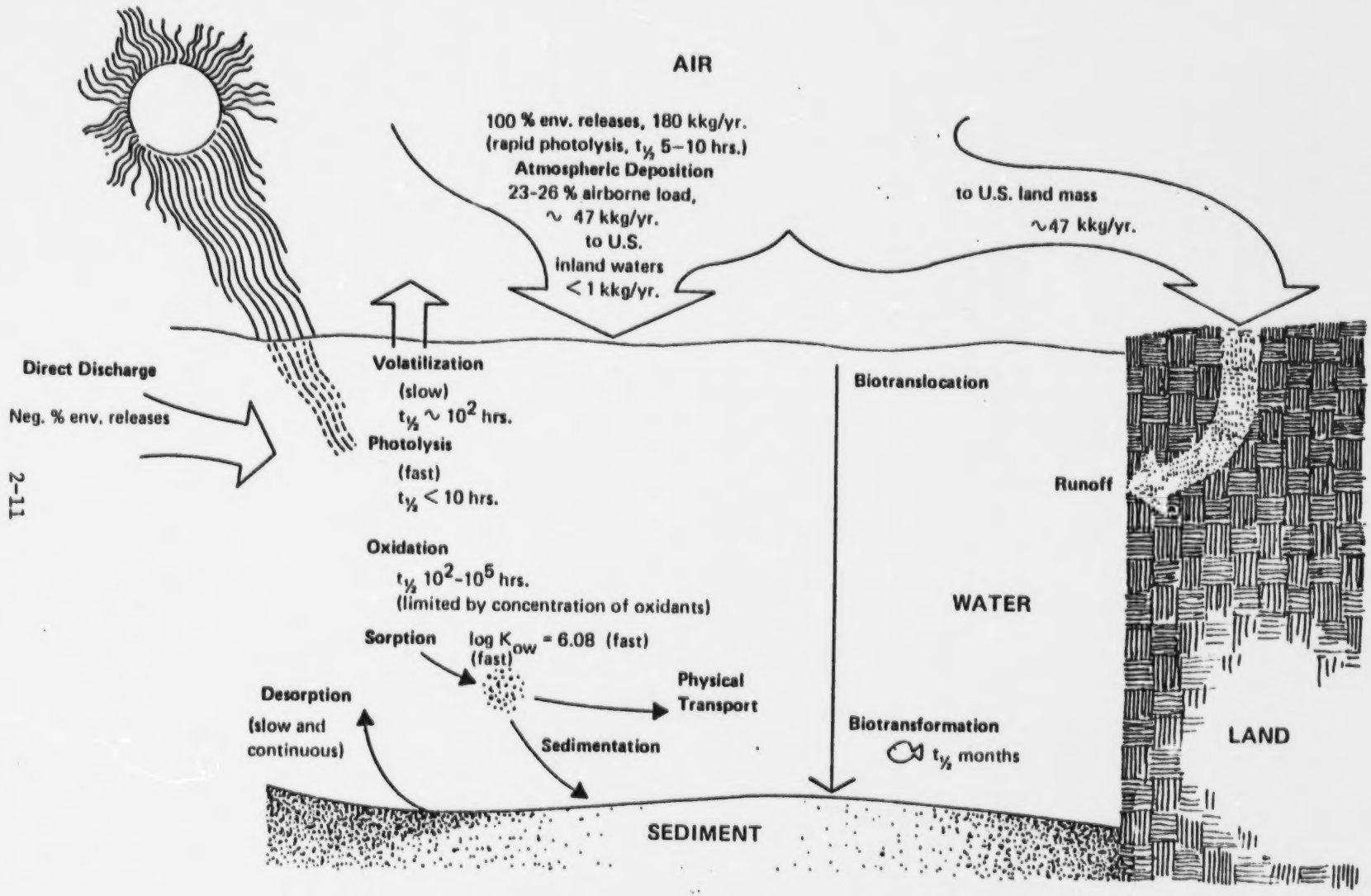

FIGURE 2-1 | SOURCES AND FATE OF BENZO[a]PYRENE IN THE AQUATIC ENVIRONMENT

Concentrations of BaP in urban air are also reported to be somewhat higher than those in rural air. Levels in U.S. cities have been reported to range from 0.13 ng/m^3 to 18.5 ng/m^3 (Colucci and Begemen 1971, Sawicki et al. 1962). In a compilation of all data available for urban air, 91% of 116 observations were less than 100 ng/m^3; in rural air, 63% of the 16 observations were less than 10 ng/m^3 (White and Vanderslice 1980).

2.1.4 Human Effects and Exposure

2.1.4.1 Human Effects

BaP has been widely tested for its carcinogenic effects. In animals it has been shown to be both a local and systemic carcinogen by oral, dermal, and intratracheal routes. The compound is also a transplacental carcinogen, an initiator of skin carcinogenesis in mice, and carcinogenic in single-dose experiments (IARC 1972; Survey of Compounds which have been Tested for Carcinogenic Activity, 1961, 1968, 1970, 1972, 1978).

In spite of the apparent availability of data on the carcinogenicity of BaP, no studies conducted to date are totally suitable for a definitive extrapolation of the carcinogenic effects of ingested BaP in humans. Two studies were chosen that represent the best available data, Neal and Rigdon (1967) and Fedorenko and Yanysheva (1966), in order to allow a quantitative estimate of risk. Several shortcomings, however, raise questions as to the validity of the extrapolated results. In both of these studies, the test populations were exposed for periods considerably less than the species lifetime. Further, tumors in both studies were limited to the forestomach; the presumably local effect of ingested BaP on rodent forestomach epithelium, in the absence of tumors at other sites, may not be relevant to humans. The site of tumor production and probable underlying mechanism are possibly unique to laboratory rodents because of the anatomy of the rodent stomach. It is possible that the forestomach tumors in mice induced by BaP may represent an unusually sensitive assay of the carcinogenicity of BaP by the oral route.

Three commonly used dose-response extrapolation models have been used to extrapolate the data from these two studies to yield estimates of carcinogenic risk of BaP to humans. The results of the extrapolation are subject to the following qualifications:

- the possible unique site and mechanism of tumor production to laboratory rodents;
- the uncertainty inherent in dose adjustment between species based upon body surface area or body weight, although body surface area was used; and

- the less-than-lifetime duration of exposure in both the studies used.

The results of extrapolating these two data sets by use of three extrapolation models are shown in Table 2-7. Due to different assumptions concerning the actual underlying mathematical relationship between dose and effect, the extrapolated risk estimated at the relatively lower exposure levels typical of actual environmental exposures varies appreciably depending upon the model and the data set used. Regardless of the model, however, higher risk estimates (by about an order of magnitude) are predicted from the data of Fedorenko and Yanysheva (1966) than from those of Neal and Rigdon (1967), possibly due to the different modes of oral administration. [Federenko and Yanysheva (1966) utilized weekly intubation, while Neal and Rigdon(1967) incorporated BaP into the diet.]

The U.S. Environmental Protection Agency (1980c) used a linear, non-threshold model and the data of Neal and Rigdon (1967) to calculate that a concentration of 2.8 ng/l in surface water would result in an estimated lifetime excess probability of cancer of 1×10^{-6} due to consumption of drinking water and fish.

The data concerning health effects other than carcinogenicity are limited. BaP appears to exert little effect on the developing embryo (Bulay and Wattenberg 1970, Rigdon and Rennels 1964). However, it is an active mutagen, inducing *in vivo* chromosomal aberrations in both hamster spermatogonia and bone marrow cells (Basler and Rohrborn 1978, Roszinsky-Kocher *et al.* 1979), and inducing positive mutagenic responses in sister chromatid exchange tests in hamsters (Bayer and Bauknecht 1977, Roszinsky-Kocher *et al.* 1979, Sirianni and Huang 1978). Because of the compound's mutagenicity, BaP exposure could also be expected to contribute to the genetic burden of a population. However, since extrapolation procedures for genetic risks are not well established, a quantitative risk assessment for these kinds of health hazards is not presently feasible.

In addition to the effects described above, the possibility exists of augmentation of effects through synergistic or co-carcinogenic mechanisms. Current understanding of these processes, however, does not allow estimation of human risk.

2.1.4.2 Human Exposure

Human exposure to BaP was evaluated considering various routes (food, drinking water, air, and smoking). These routes were considered as quantitatively as possible, even though data are often scarce.

Smoking appears to be an important route of exposure. Levels of 0.025 µg BaP per cigarette in mainstream smoke were reported by Schmeltz *et al.* (1975). At these levels, smokers could be exposed to 0.025-2.5 µg/day, depending upon the type and number of cigarette smoked, the

TABLE 2-7. ESTIMATED LIFETIME EXCESS PROBABILITY OF CANCER TO HUMANS DUE TO BENZO[a]PYRENE INGESTION AT VARIOUS EXPOSURE LEVELS BASED ON THREE EXTRAPOLATION MODELS[a]

Exposure Level (μg/day):	Estimated Lifetime Excess Probability of Cancer at Indicated Exposure Level					
	0.001	0.01	0.1	1	10	100
Extrapolation Model						
Linear Model						
Neal and Rigdon data	1×10^{-6}	1×10^{-5}	1×10^{-4}	1×10^{-3}	1×10^{-2}	9.2×10^{-2}
Fedorenko and Yanysheva data	1.1×10^{-5}	1.1×10^{-4}	1.1×10^{-3}	1.1×10^{-2}	1.1×10^{-1}	6.7×10^{-1}
Log-probit Model						
Neal and Rigdon data	1×10^{-9}	1×10^{-7}	1.4×10^{-5}	7.5×10^{-4}	1.5×10^{-2}	1.2×10^{-1}
Fedorenko and Yanysheva data	3×10^{-7}	2.6×10^{-5}	1.2×10^{-3}	2.1×10^{-2}	1.5×10^{-1}	4.8×10^{-1}
Multi-stage Model						
Neal and Rigdon data	1.7×10^{-6}	1.7×10^{-5}	1.7×10^{-4}	1.7×10^{-3}	1.6×10^{-2}	1.5×10^{-1}
Fedorenko and Yanysheva data	7×10^{-6}	7×10^{-5}	6.8×10^{-4}	6.8×10^{-3}	6.5×10^{-2}	4.8×10^{-1}

[a]The lifetime excess probability of cancer represents the increase in probability of cancer over the normal background incidence, assuming that an individual is continuously exposed to BaP at the indicated daily intake over a 70-year lifetime. There is considerable variation in the estimated risk due to uncertainty introduced by the use of laboratory rodent data, by the conversion to equivalent human dosage, and by the application of hypothetical dose-response curves. In view of several conservative assumptions that were utilized, it is likely that these predictions overestimate the actual risk to humans.

amount of smoke inhaled, and the number of cigarettes smoked. An estimated 54.1 million persons in the U.S. smoke cigarettes, and of these 25-30% smoke more than 25 cigarettes per day (U.S. DHEW 1979). Thus a large segment of the population (those smoking >25 cigarettes per day) could be exposed to BaP in the 0.6-2.5 µg/day range resulting from mainstream smoke alone.

Although levels of BaP in smoke-filled rooms have not been measured specifically, estimates were made based upon CO levels summarized by Burns (1975). CO levels of 44-92 mg/m^3 were reported for rooms (38-92 m^3) where 30-80 cigarettes had been smoked with no ventilation. BaP levels of about 0.07-0.2 µg/m^3 were calculated for a small room with no ventilation, based upon a CO/BaP ratio of $\frac{50,000}{0.085}$ in side-stream smoke. A non-smoker exposed to such a situation 2 hours/day would be exposed to about 0.25-0.7 µg BaP/day; however, there are numerous uncertainties in these calculations.

Levels of BaP in raw foods are generally low. Higher levels, however, result from cooking processes, especially charcoal broiling and smoking. Table 2-8 shows daily exposure due to consumption of such foods and summarizes the assumptions made in developing the estimates. The estimated "typical exposure" via food ingestion is about 0.05 µg/day. Consumption of large amounts of charcoal-broiled food could result in exposure amounting to as much as 6 µg/day.

The levels reported by White and Vanderslice (1980) for ambient air imply that persons in urban areas are exposed to 0.02-2 µg/day, while persons in rural areas are generally exposed to 0.0002-0.2 µg/day, assuming respiratory flow of 20 m^3/day. Although woodburning has been shown to increase indoor air levels over outdoor air, these levels (mean of about 5 ng/m^3) still fall within the range for rural and urban areas (Moschandreas et al. 1980). The U.S. EPA (1978b) has estimated the population sizes exposed to various concentrations. These results shown in Table 2-9 suggest that most individuals in the U.S. are exposed to BaP by inhalation of ambient air in the range of 0.02-0.1 µg/day.

Drinking water does not generally appear to represent a significant source of exposure to BaP. On the basis of the limited data of Basu and Saxena (1978), a typical exposure of 0.0006 µg/day, and a maximum of 0.004 µg/day were calculated for drinking water, assuming a consumption of 2 l/day.

A summary of all exposure pathway estimates appears in Table 2-1.

2.1.5 Aquatic Effects and Exposure

No data from acute toxicity bioassays were available for BaP. However, tissue damage, abnormal growth, and the production of cancer-like growths have been reported. Teratogenic and mutagenic effects have

TABLE 2-8. LEVELS OF BENZO[a] PYRENE GROUP PAHs IN FOOD AND ESTIMATED INGESTION EXPOSURE

		Consumption (g/day)		Benzo[a]pyrene Contamination (µg/kg)		Benzo[a]pyrene Intake (µg/day)	
		Typical	Max.	Typical	Max.	Typical	Max.
Charcoal broiled beef[a]							
Hamburger		10	NA	NA	2.6	NA	0.03
Steak		3	86	5	50	0.02	4.3
Smoked pork[b]		1	27	2	55	0.002	1.5
Smoked sausage[c]		1.5	30	NA	4	0.006	0.12
Smoked fish[d]		0.1	14	1	37	0.0001	0.5
Oil		18	NA	1	8	0.02	0.14
Fruits		205	NA	0.02	6	0.004	1.2
Grains		256	NA	NA	0.3	NA	0.08
Vegetables	Total	248	NA	0.01	0.1	0.002	0.2
	Leafy	40	NA	NA	7.5	NA	0.3

[a] Consumption of beef - 86 g/day, 15% charcoal-broiled - 80% hamburger, 20% steak. Worst case maximum 86 g consumption of charcoal-broiled steak.

[b] Consumption of pork - 27 g/day, 5% smoked. Worst case maximum, 27 g/day smoked.

[c] Consumption of sausage - 30 g/day, 5% smoked. Worst case maximum 30 g/day smoked.

[d] Consumption of fish - 14 g/day, 1% smoked. Worst case maximum, 14 g/day smoked.

Source: USDA (1978, 1980), U.S. EPA (1980), White and Vanderslice (1980).

TABLE 2-9. ESTIMATED SIZE OF THE U.S. POPULATION EXPOSED TO RANGES OF BENZO[a]PYRENE CONCENTRATIONS IN AMBIENT AIR

	BaP Concentration (ng/m^3)			
	<0.5	0.5-1.0	1.0-5.0	>5.0
Population (1000's) Exposed	73,294	26,731	102,132	1059

Note: For some locations for which monitoring data were unavailable (representing about 50% of the population) the upper limits of 95% confidence intervals of national average concentrations were used. These levels were as follows:

urban SMSA[a]	1.3 ng/m^3
urban non-SMSA	1.4 ng/m^3
rural	0.23 ng/m^3

[a]Standard Metropolitan Statistical Area

Source: U.S. EPA (1978b).

also been observed in aquatic invertebrates and lower vertebrates (Korotkova and Tokin 1968, Neff 1979).

The available data on BaP concentrations in ambient waters suggest that exposure of aquatic organisms is low. As discussed previously, levels in surface water of less than 1 µg/l are most common. The levels of BaP in sediments may frequently exceed 1000 µg/kg, and represent a potential source of exposure. However, the bioavailability of sediment-bound BaP is unknown.

2.1.6 Risk Considerations

2.1.6.1 Human Risk of Carcinogenicity

The results of the extrapolation of the animal studies on carcinogenicity have been discussed and presented previously in Table 2-7 (p. 2-14). The qualifications and major assumptions have also been discussed and underline the caution with which these calculations must be viewed.

The risks based upon these calculations and the exposure estimates made for specific subpopulations are shown in Table 2-1 (p. 2-2). The highest estimated excess probability of cancer appears to be for smokers, especially heavy smokers with an estimated incidence of 230-7700 excess cancers/year. Dietary exposure, primarily due to smoked and charcoal-broiled foods, may result in an estimated 13-1900 excess cancers/year. Risk estimates associated with inhalation exposures show a wide range. Urban residents may experience 1-95,000 excess cancers/year and residents of rural areas, << 1 to 2400 excess cancers/year. Drinking water consumption generally appears to present a lower degree of risk. It should be noted, however, that these estimates are based upon numerous assumptions and limited monitoring data. These limitations are discussed in this summary and Volume IV of this report.

2.1.6.2 Human Risk Associated With Non-Carcinogenic Effects

Little information exists regarding other potential adverse effects of exposure to BaP. Although this compound has been identified as an active mutagen in numerous systems, a quantitative estimate of genetic risk is not feasible. In addition, one should not overlook possible augmentation of risk through synergistic or co-carcinogenic mechanisms; however, current understanding of the co-carcinogenesis process is not sufficiently adequate to allow estimation of associated human risks at this time.

2.1.6.3 Risk to Aquatic Organisms

Data on effects of BaP on biota are extremely limited. As a result, no statement of potential risk, even qualitative, can be made at this time. Similarly, the U.S. EPA (1980c) was unable to set water quality criteria for BaP due to the lack of data. It should be noted, however, that high sediment levels may pose some risk to aquatic biota,

although there is at present no way to evaluate the environmental risks of contaminated sediment.

2.2 NAPHTHALENE

2.2.1 Risk Conclusions

A review of the environmental monitoring and human effects data indicates that the dose levels at which acute effects of naphthalene have been observed in humans or mammals are generally more than five orders of magnitude greater than human exposure levels through ingestion, and at least three orders of magnitude higher than inhalation exposure levels to specific subpopulations associated with mothball usage and cigarette smoking. Chronic toxicity studies have failed to demonstrate the carcinogenic activity of naphthalene. The major effects of naphthalene on humans include cataracts and hemolytic anemia. There appears to be little acute risk to humans from environmental exposure to naphthalene; however, severe adverse effects are possible from accidental ingestion of substantial quantities of naphthalene.[3]

The U.S. EPA has not established a water quality criterion for the protection of human health due to the inadequacy of the data on the chronic toxicity of naphthalene and the lack of epidemiologic studies (U.S. EPA 1980d).

There is very little information available on the extent of exposure of aquatic biota to naphthalene. However, the available monitoring data suggest concentrations reported in ambient water are about two orders of magnitude lower than the levels associated with acute or chronic effects to aquatic organisms.[4]

2.2.2 Releases of Naphthalene to the Environment

Production of naphthalene in 1978 totaled 2.35×10^5 kkg, 70% from coal tar and 30% from petroleum (Abshire *et al.*, 1980). Total environmental releases in 1978 were equivalent to 5% of production: 10,600 kkg were released to air, 300 kkg were released to land, 240 kkg were released to POTWs, and approximately 340 kkg were discharged directly to surface waters. Production activities accounted for the estimated annual release of 83 kkg (Brown 1975); direct uses of naphthalene released 5,000 kkg, and sources of inadvertently released naphthalene total more than 6,000 kkg.[5]

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[3]These conclusions are taken from Volume II of this report.
[4]See footnote 3.
[5]These estimates were developed by Acurex, Inc. for EPA under Contract No. 68-01-6017. Their data are summarized in Volume II of this report.

The major aquatic releases of naphthalene arise from coal tar production and distillation; minor releases include naphthalene production, oil spills, and POTW releases. Combustion sources account for most of the annual atmospheric emissions of naphthalene (5,100 kkg), largely due to residential wood and coal heating; moth repellants account for the next largest atmospheric release, 4,700 kkg.[6]

2.2.3 Fate and Distribution in the Environment

Most of the environmental releases of naphthalene are to the atmosphere. Atmospheric deposition (wet and/or dry fallout) is expected to remove only 2-3% (200-300 kkg on the basis of 1978 data) of the total atmospheric load. This estimate was developed using the techniques described in Arthur D. Little, Inc. (1981). It is estimated that the amount deposited directly onto U.S. inland surface waters will be proportional to their area, i.e., 2% of the total continental United States (U.S. Bureau of the Census 1980). Therefore, ∿5 kkg can be expected to fall on inland surface waters of the U.S. annually; only a fraction of the fallout to land will ultimately reach aquatic systems via runoff and leaching. The naphthalene remaining in the atmosphere is expected to undergo rapid photo-oxidation to quinones and other oxygenated compounds (Radding et al. 1976); however, a thorough review of the atmospheric fate pathways is beyond the scope of this report.

Since naphthalene has a vapor pressure of 0.09 torr at 25°C (SRI 1980), and log of the octanol:water partition coefficient of 3.37 (Versar, Inc. 1979), a large amount of the naphthalene in surface waters can be expected to be removed to the atmosphere via volatilization; removal to the sediment via adsorption and sedimentation may be important if there is a significant amount of suspended material in the aquatic system (Neff 1979). Biodegradation may also be a significant fate process for naphthalene in aquatic systems (Schwall and Herbes 1978). The application of the Mackay equilibrium partitioning model (Mackay 1979) confirms the prediction that large amounts of the aquatic naphthalene will be removed to the atmosphere and sediment. Calculations based on the Exposure Analysis Modeling Systenm [EXAMS] (U.S. EPA 1980b) also predict that biotransformation, volatilization, photolysis, and physical transport could all be important fate processes, depending upon the actual conditions in the environment. In comparison with the other PAHs, photolysis, oxidation, and sedimentation of naphthalene are much less important pathways; volatilization is the most important fate process in static aquatic systems (Smith et al. 1978, Radding et al. 1976, Zepp and Schlotzhauer 1979). The extent of volatilization depends upon temperature, wind velocity, and current velocity. In rivers, the major fate pathway is transport of adsorbed naphthalene to the oceans (White and Vanderslice 1980).

[6]See footnote 5.

EXAMS results for six generalized aquatic systems suggest that the naphthalene in sediment will tend to remain there unless there is considerable mixing between the sediment and the water column, since neither photolysis nor anaerobic degradation are important fate pathways. In summary, although the sediments and the water columns of aquatic systems may contain naphthalene, accumulation will occur in the sediment.

Monitoring data confirm that concentrations in sediment are higher than those in water. The STORET data base (U.S. EPA 1980e) reports actual measurements of naphthalene in ambient waters ranging from 0.005 µg/l to 17 µg/l; effluent concentrations up to 36,000 µg/l are reported. Actual sediment concentrations reported in STORET range from 0.02 µg/kg to 496 µg/kg. Drinking water concentrations up to 1.4 µg/l were reported in the literature (U.S. EPA 1980d). Ambient air concentrations of naphthalene were reported to be 0.35 ng/m^3 in an urban area, and 0.05 ng/m^3 in a rural area (Krstulovic et al. 1977). Concentrations to which industrial workers may be exposed range from 10^2 ng/m^3 to 10^6 ng/m^3 in the vapor phase, and up to 4 x 10^3 ng/m^3 adsorbed onto particulate matter (Bjorseth et al. 1978a,b).

2.2.4 Human Effects and Exposure

Naphthalene is absorbed through ingestion, inhalation, and skin contact; however, the rate and extent of absorption and the tissue distribution have not been studied in detail (U.S. EPA 1980d). There is little quantitative information on the carcinogenic or long-term effects of naphthalene. Two experiments by Knake (1956) indicated a slight but not statistically significant increase in lymphosarcomas in rats and lymphocytic leukemia in mice; several other skin painting studies were negative for carcinogenesis. Mutagenicity studies were also negative (U.S. EPA 1980d). A single study noted retarded heart development and cranial ossification in rats exposed to high concentrations throughout gestation (Harris et al. 1979).

The major effects linked to naphthalene exposure include cataracts, sometimes accompanied by retinopathy and hemolytic anemia. Near blindness was reported in one human upon ingestion of 5 g naphthalene (~70 mg/kg); bilateral cataracts were also reported among workers exposed to naphthalene, although details of that study were not available (Ghetti and Mariani 1956). Cataracts and retinopathy were reported in laboratory animals at dose levels of 1,000 mg/kg/day. The oral LDLo (lowest lethal dose) reported for naphthalene in children was ~100 mg/kg (Sax 1979); the oral LD_{50} (lethal dose to 50% of the subjects) observed in rodents was ~2,000 mg/kg (U.S. EPA 1980d).

Individuals with relative deficiencies in the enzymes (G6PD) needed to maintain glutathione levels in red blood cells (approximately 100 million people worldwide) may have increased sensitivity to naphthalene (Wintrobe et al. 1974); the fetus and the newborn may also have an

increased sensitivity (Dawson et al. 1958). These subpopulations are at increased risk of developing hemolytic anemia, which may lead to renal damage.

Data on human exposure to naphthalene are limited. The maximum naphthalene concentration reported in drinking water of 1.4 µg/l (U.S. EPA Region IV, unpublished data) corresponds to a daily intake level of 2.8 µg/day, assuming ingestion of 2 liters of water/day. Ingestion of fish could lead to intake of another 0.4 µg/day on the basis of a bioconcentration factor of 10.5, an ambient water concentration of 2 µg/l, and a fish consumption of 21 g/day (U.S. EPA 1980d,e; USDA 1978). Data concerning naphthalene concentrations in food were not available.

Exposure estimates for inhalation of ambient air are quite low, 0.001 µg/day to 0.007 µg/day based upon the monitoring data of Krstulovic et al (1977) and assuming a respiratory flow of 20 m^3/day. However, smoking and mothball usage could increase inhalation exposure levels. The naphthalene content in mainstream smoke from 85 mm cigarettes was shown to be 3 µg/cigarette (Schmeltz et al. 1975). Since about one-third of U.S. adults smoke cigarettes, and 25-30% of these smoke more than 25 cigarettes per day (U.S. DHEW 1979), a large segment of the U.S. population (~15 million people) could be exposed to naphthalene doses greater than 75 µg/day via this exposure route. Naphthalene content in sidestream smoke was reported to be 46 µg/cigarette by Schmeltz et al. (1976); in a smoke-filled room, the naphthalene concentration was estimated (by analogy to carbon monoxide measurements) to be as high as 83 µg/m^3. At an assumed respiratory flow of 1.8 m^3/hr, even a nonsmoker exposed to these levels for 2 hours/day could receive up to 300 µg/day. Smokers in the same environment would receive doses associated with both the mainstream and sidestream smoke.

Use of mothballs containing naphthalene may result in increased indoor air concentrations. It was estimated that inhalation exposures related to domestic use of mothballs may be as high as 140 µg/day by analogy to dichlorobenzene levels reported by Morita and Ohi (1975). Approximately 26 x 10^6 households in the U.S. use mothballs, though only one-third of the mothballs produced contain naphthalene (U.S. EPA 1980h).

2.2.5 Aquatic Effects and Exposure

Naphthalene concentrations at which acute toxic effects have been reported for aquatic organisms range from 0.92 mg/l (Thomas and Rice 1974) to 199 mg/l (Legore 1974). Certain sublethal effects (behavioral) on aquatic organisms have been observed at concentrations as low as 0.12 mg/l (Pearson and Olla 1979); histopathologic changes were noted in estuarine fish at concentrations from 0.002 mg/l to 0.02 mg/l (Neff 1979). A chronic effects value of 0.62 mg/l (U.S. EPA 1980d) was obtained in an embryo-larva test conducted on fathead minnows. Monitoring data for naphthalene indicate that the levels in ambient waters are

generally about 10 µg/l; sediment levels range from 0.02 µg/kg to less than 500 µg/kg (U.S. EPA 1980e).

2.3 ANTHRACENE, ACENAPHTHENE, FLUORANTHENE, FLUORENE, PHENANTHRENE, AND PYRENE

2.3.1 Risk Conclusions

The human effects data for this group of PAHs are inadequate to warrant a quantitative extrapolation of the human risk from environmental exposure to these compounds. There are virtually no toxicological data. None of the compounds in this group has been reported to be carcinogenic by the oral route (Schmahl 1955, Innes et al. 1969); pyrene and fluoranthene, however, have been reported to be co-carcinogens (Salaman and Roe 1956, Scribner 1973). Due to inhalation of mainstream cigarette smoke reported to contain fluoranthene and fluorene (Schmeltz et al. 1975), smokers may be exposed to higher levels of these PAHs than is the general population. However, the limits on the risk associated with these environmental exposure scenarios cannot be defined at this time.

The U.S. EPA reports that sufficient data on acenaphthene were not available to permit the derivation of a water quality criterion that would protect humans against the potential toxicity of this compound. However, the level for controlling undesirable odor and taste quality in ambient water has been estimated to be 20 µg/l on the basis of organoleptic data which have no demonstrated relationship to adverse human health effects (U.S. EPA 1980f). For fluoranthene, an ambient water quality criterion for the protection of human health has been set at 42 µg/l (U.S. EPA 1980g). This criterion was based on a no-effect level for mortality in a mouse skin-painting experiment. Ambient surface water concentrations above these recommended levels have been reported for both of these compounds. No specific water quality criteria have been set for the other PAHs in this group (U.S. EPA 1980c).

Risk to aquatic biota exposed to ambient concentrations of these PAHs is expected to be low. The U.S. EPA has not established ambient water quality criteria for these compounds for the protection of aquatic life (U.S. EPA 1980c,f,g). However, all ambient concentrations in the STORET data base (U.S. EPA 1980e) were below the levels that were reported to be acutely toxic to freshwater organisms. The STORET concentration data for surface water do overlap the range of chronic and acute toxic effects levels for marine organisms, which appear to be more sensitive to these PAHs (Neff 1979, U.S. EPA 1980g); however, there are no monitoring data specifically for marine systems. Since the potential does exist for bioaccumulation of these PAHs in zooplankton (Giddings et al. 1978), and subsequent biomagnification by fish (Herbes 1976), direct comparison of ambient concentrations with effects levels may not adequately describe the risk to aquatic organisms.

2.3.2 Releases to the Environment

The six PAHs in the anthracene group are all used commercially. Total use in 1979 amounted to approximately 830 kkg; most (720 kkg) was imported. The annual (domestic and imported) supply of fluorene and phenanthrene was reported to be less than 1 kkg of each (U.S. Dept. of Commerce 1980).

These PAHs are used as intermediates in the synthesis of a variety of compounds, including pharmaceuticals, pigments, plastics, pesticides, and photographic chemicals. Since the PAH compounds are consumed in these processes, a small, approximately 1%, release to the environment may be assumed; thus, about 8 kkg of these six PAHs were estimated to be released in 1979, distributed almost equally among the air, land, and water compartments.[7]

The chief source of inadvertent releases of anthracene and the other PAHs in this group is combustion. Burning of wood for residential heating generates the largest amount of these PAHs, 3,200 kkg or 60% of the total (5,400 kkg) estimated annual atmospheric emissions of anthracene group PAHs; coal, gas, and oil fired residential heating units release less than 800 kkg combined. Anthracene and phenanthrene account for 75% of the total atmospheric emissions from residential wood burning attributable to the PAHs in this group. Other combustion sources of these PAHs include agricultural burning, prescribed burning, wildfires, and combustion of gasoline.[8]

In 1978, coal tar distillation resulted in the inadvertent production of approximately 175,000 kkg of anthracene group PAHs (U.S. DOE 1979, Rhodes 1954). Of this total, the amounts released to the environment are estimated to be: 30 kkg to air, 14 kkg to land, 17 kkg to POTWs, 22 kkg to surface water, for a total estimated environmental release of 83 kkg in 1978. Approximately 15 kkg of the PAHs in this group were released to water in oil spills and 14 kkg to air in petroleum refinery wastestreams, according to 1978 data.[9]

The total environmental releases of the anthracene group PAHs for 1978 are estimated to be about 5,600 kkg; of this total, 5,400 kkg or 97% is estimated to be released to air, 60 kkg to surface water, 100 kkg to POTWs, and approximately 1 kkg to land.[10]

2.3.3 Fate and Distribution in the Environment

The environmental release data indicate that most discharges of anthracene and the other PAHs in this group are to the atmosphere.

[7]These estimates were developed by Acurex, Inc. for EPA under Contract 68-01-6017. Their data are summarized in Volume III of this report.
[8]See footnote 7.
[9]See footnote 7.
[10]See footnote 7.

Atmospheric deposition (from both wet and dry processes) may remove 5-26% of the atmospheric load of anthracene in urban areas, accounting for 75-300 kkg/yr of anthracene fallout. It is estimated that about 2% will fall directly on inland surface waters, representing 1.5-7-8 kkg/yr anthracene. The upper limit on this range corresponds to deposition in areas near combustion sources, where the anthracene will be primarily adsorbed onto particulates. The percentage of these PAHs that remains in the atmosphere will be degraded by photolysis to oxygenated compounds, including quinones (Radding et al. 1976).

Since the water solubilities of the anthracene group PAHs are relatively low and the octanol:water partition coefficients are fairly high, adsorption onto both organic and inorganic matter is a primary removal pathway for these compounds in the water column. The particulate matter will ultimately be transported to the sediment where these PAHs will accumulate (Neff 1979, Smith et al. 1978); biodegradation and photo-oxidation in sediments are expected to be quite slow. The fraction of these PAH that remain in the water column is expected to be degraded photolytically (Zepp and Schlotzhauer 1979, Radding et al. 1976); however, the extent of this removal pathway will be affected by the turbidity and light penetration in the actual system (Southworth 1977). Volatilization from water is not expected to be a major fate process, but the relative importance of this pathway differs among aquatic systems (Southworth 1979). Bioconcentration factors for anthracene are on the order of several hundred (Herbes 1976); half-lives for biodegradation have been determined to be 1-2 weeks in acclimated cultures (Quave et al. 1980).

EXAMS (U.S. EPA 1980b) calculations for anthracene indicate that in all model systems, except the oligotrophic lake where sedimentation rates are low, over 80% of the anthracene resides in the sediment compartment when the system is at steady state. Rapid photolysis is predicted by EXAMS for the anthracene remaining in the water column of clear, quiescent systems; volatilization is important in the pond and eutrophic lake systems where light penetration is reduced by suspended matter. Biological degradation is important only in the highly productive eutrophic lake. In the more dynamic river systems, physical transport (downstream) accounts for most of the anthracene removal.

Anthracene and the related PAHs have been detected in all environmental media (Kim and Stone 1979; U.S. EPA 1980c,e,f,g; White and Vanderslice 1980). Monitoring data support the predictions that significant amounts of anthracene and related PAHs will reside in the sediments. The majority of the STORET surface water concentrations of these PAHs are less than 100 µg/l; STORET effluent data include concentrations ranging from <1 µg/l to <1,000 µg/l for these PAHS (U.S. EPA 1980e). Various other sources report effluent and sewage concentrations of fluoranthene from 2 µg/l to 20 µg/l (U.S. EPA 1980g). Levels of pyrene and fluoranthene in soil were high, up to 120,000 µg/kg (White and Vanderslice 1980). Concentrations of pyrene in several edible marine species were reported to range from less than 0.6 µg/kg to

58 µg/kg (wet weight) (Pancirov and Brown 1977). PAH levels in air from a variety of urban locations were generally less than 12 ng/m^3 (U.S. EPA 1980c,g).

2.3.4 Human Effects and Exposure

The human effects data for anthracene and the other PAHs in this group are very limited. There are essentially no toxicological data from which to determine the dose associated with acute toxic effects in humans or laboratory mammals. The chronic effects of these compounds have not been studied extensively; however, the available data indicate that none has been shown to be carcinogenic by the oral route (Schmahl 1955). Phenanthrene and pyrene show weak tumor-initiation activity in mouse skin carcinogenesis experiments (U.S. EPA 1980c,g). Mutagenicity studies were negative, and no data were available on the teratogenic effects of these six compounds. Since all six PAHs in this group are lipid-soluble, absorption and distribution throughout the body are expected to occur.

On the basis of available monitoring data, the typical environmental exposure from ingestion of food and water and inhalation of ambient air is estimated to be less than 1 µg/day for each of these six PAHs; maximum exposure levels could be as high as 21 µg/day for fluoranthene.[11] However, very limited data are available, particularly for drinking water. Consumption of food, particularly charcoal-broiled meats, appears to be the major route of exposure for all of the six PAHs in this group (U.S. DOA 1978, 1980, U.S. EPA 1980c,f,g, White and Vanderslice 1980). Fluoranthene and pyrene appear to have the greatest potential for human exposure from environmental sources.

A significant percentage of the U.S. population may be exposed to higher levels of these PAHs through inhalation of cigarette smoke; 5.2 µg fluoranthene and 8.4 ug fluorene have been reported in the mainstream smoke of one pack of cigarettes (Hoffman et al. 1972). The exposure of this subpopulation of smokers to these PAHs may be greater than the typical exposure to the general population.

2.3.5 Aquatic Effects and Exposure

The levels of these PAHs at which acute toxic effects were observed in freshwater organisms range from 1.7 mg/l to 325 mg/l (U.S. EPA 1978c). Marine organisms appear to be much more sensitive, displaying acute effects at concentrations of 40 µg/l fluoranthene and 150 µg/l phenanthrene (U.S. EPA 1978c). With respect to fish and invertebrates, fluoranthene appears to be the most toxic of the six PAHs in this group; however, plants were found to be very resistant to fluoranthene (U.S. EPA 1978c). Chronic effects in marine species have been associated with

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[11]Data taken from Volume III of this report.

fluoranthene concentrations as low as 12 µg/l (U.S. EPA 1978c). Microcosm experiments conducted in the presence of sunlight have shown that 1 µg/l to 10 µg/l of anthracene killed all organisms present (Personal Communication, P. Landrum et al., Savannah River Ecology Laboratory 1980). However, these laboratory data may not be directly representative of the effects of these PAHs in actual environmental systems.

The monitoring data documenting the exposure of aquatic biota to these PAHs are quite limited. All of the ambient water concentrations of anthracene, and most of those for the other PAHs in this group, were reported to be less than 100 µg/l (U.S. EPA 19890e); all of the actual concentrations in ambient waters were reported to be below 1.7 mg/l, the lowest acute effects level for freshwater organisms (U.S. EPA 1978c). However, 25-40% of the detectable concentrations for five PAHs in this group (excluding anthracene) were between 100 µg/l and 1,000 µg/l; only 8-17 observations above detection limits were reported (U.S. EPA 1980e). Acute toxic effects and chronic effects for marine organisms have been attributed to concentrations within this range. Sediment concentrations of acenaphthene and fluorene were reported to be within the range of 2 µg/kg to 50 µg/kg; unremarked sediment concentrations for the other PAHs ranged from not detected to 1,000 µg/kg (U.S. EPA 1980e). The extent of the bioavailability of these compounds bound to sediments has not been clearly determined at this time.

2.4 ACENAPHTHYLENE, BENZ[a]ANTHRACENE, BENZO[b]FLUORANTHENE, BENZO[k]FLUORANTHENE, BENZO[g,h,i]PERYLENE, CHRYSENE, DIBENZ[a,h]ANTHRACENE, AND INDENO[1,2,3-c,d]PYRENE

2.4.1 Risk Conclusions

Data for the PAHs included in this group were inadequate for purposes of quantitative risk assessments since dose-response data were unavailable. Benz[a]anthracene and dibenz[a,h]anthracene are both carcinogenic in mice by the oral route (IARC 1972). The compounds are also complete carcinogens in mice exposed by the dermal route, as are benzo[b]fluoranthene and indeno[1,2,3-c,d]pyrene. Benzo[g,h,i]perylene is a co-carcinogen with BaP and benz[a]anthracene; benzo[b]fluoranthene, chrysene, and indeno[1,2,3-c,d]pyrene are all initiators of skin carcinogenesis in mice. No carcinogenicity data were found for acenaphthylene (U.S. EPA 1980c, IARC 1972, Habs et al. 1980).

Only limited data are available describing the acute and chronic effects of the PAHs in this group for aquatic biota. The results of the only reported laboratory toxicity test for any of these compounds indicate that benz[a]anthracene at a concentration of 1 µg/l caused 87% mortality in bluegill sunfish in 6 months (Brown et al. 1975). Levels of these compounds in ambient surface water are generally lower than 1 µg/l, although levels up to about 2000 µg/l have been reported (U.S. EPA 1980c,e). High sediment levels of PAHs, as may be found near industrialized areas, may pose some risk to aquatic biota, although the extent of bioavailability of sediment-bound PAH is not well documented.

2.4.2 Releases to the Environment

The PAHs in this group are released primarily to the atmosphere and result mainly from combustion sources. Residential heating accounts for the largest releases, since these emissions are largely uncontrolled. Emission factors indicate that fireplace burning produces a smaller amount of PAHs per unit of fuel than do the more airtight wood-burning units used for primary heating. On the basis of emission factors and quantity of fuel consumed in 1978, the following estimates of annual atmospheric releases for this group PAHs can be made:

- residential coal combustion -- 130 kkg;
- fireplace burning, primary wood heating and auxiliary wood heating -- 40 kkg, 630 kkg, and 800 kkg, respectively;
- two types of forest fires, prescribed burning and wildfire burning -- 450 kkg and 260 kkg, respectively; and
- agricultural burning -- 170 kkg.

With the exception of gasoline combustion (accounting for the release of about 40 kkg of this group of PAHs in 1978), all other combustion sources are estimated to release total quantities of these PAHs that are much smaller on a national level than the amounts released during combustion of coal and wood. Utility boilers, municipal and commercial incinerators, and industrial internal-combustion engines are all controlled units, have relatively high combustion efficiencies, and are believed to release relatively low amounts of these PAHs.[12]

In addition to combustion sources, these PAHs are released from a few contained sources, primarily related to coal tar and petroleum. Coal tar production and distillation activities account for the environmental release of approximately 50 kkg (1978) of these six PAHs; their distribution is believed to be almost equally divided among releases to air, land, surface water, and POTWs. Significant environmental releases associated with petroleum sources (exclusive of gasoline combustion) are believed to be limited to oil spills and atmospheric releases at refineries.[13]

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[12]These estimates were developed by Acurex, Inc. for EPA Under Contract No. 68-01-6017. These data are summarized in Volume IV of this report.

[13]See footnote 12.

2.4.3 Fate and Distribution in the Environment

The PAHs in this group are released to the environment primarily as products of combustion. Although photolysis of atmospheric PAHs is relatively rapid (Versar 1979), wet and dry deposition of the atmospheric PAH emissions probably represent the major input pathways to the aquatic environment based upon the analysis conducted for BaP described previously in this summary and in Volume IV of this report.

Calculations using the model of Mackay (1979) indicate that at equilibrium 99.9% of BaP in the aquatic environment resides in the sediment compartment; very small amounts will be found in the air (vapor phase BaP), water, or biota. This distribution of BaP is probably representative of the fate of other compounds in this group as well. The U.S. EPA (1980b) EXAMS model was used to predict the fate and distribution of BaP in six generalized aquatic systems. The results indicate that in all of the systems modelled, maximum BaP concentrations in sediment can be expected to be higher than total water concentrations by factors up to 10^4. The EXAMS data also indicate that more than 90% of the BaP will reside in the sediment for all aquatic systems examined, with the exception of the oligotrophic lake (81%). Again, this pattern of distribution should hold true for other compounds in this group of PAHs.

The most significant fate processes for these compounds in aquatic systems include adsorption (with subsequent transport to the sediment) and chemical degradation (photolysis). Volatilization and biodegradation are expected to be slow processes for the PAHs in this group (Smith et al. 1978, Quave et al. 1980, Colwell and Sayler 1978, Gardner et al. 1979). These compounds are expected to be relatively persistent in aquatic systems as was predicted for BaP by use of EXAMS.

There are fewer monitoring data for the other PAHs in this group than for BaP. Most of the observations in the STORET data base for the PAHs in this group are reported to be below the detection limits. The PAH concentrations reported above detection limits for sediment samples range from 0.002 µg/kg to 2600 µg/kg. Detectable ambient water concentrations recorded in STORET range from 0.01 µg/l to 1500 µg/l (U.S. EPA 1980c).

Data from other sources indicate that the concentrations of these compounds in ambient waters are generally less than 1 µg/l (White and Vanderslice 1980). Concentrations in drinking water were reported to range from 0.1 ng/l to 4.0 ng/l (Basu and Saxena 1978). In this survey, the highest concentration for the PAHs in the BaP group was 4.0 ng/l, reported for benzo[g,h,i]perylene in a drinking water sample taken from Philadelphia, PA.

Ambient air concentrations of these PAHs were also reported to be significantly higher in urban areas than in rural areas. In a study of

seven urban areas, the highest PAH concentrations in air (up to 21.3 ng/m^3 for benzo[g,h,i]perylene) were reported for Detroit, MI (Sawicki et al. 1962). Data presented in a separate review of ambient levels of PAHs indicate that most rural air concentrations are less than 10 ng/m^3; urban air concentrations up to 100 ng/m^3 were reported for many of these PAHs (White and Vanderslice 1980).

2.4.4 Human Effects and Exposure

Among the compounds in this group, benz[a]anthracene, dibenz[a,h]anthracene, benzo[b]fluoranthene, and indeno[1,2,3-c,d]pyrene are complete carcinogens for mouse skin; benzo[g,h,i]perylene is a co-carcinogen with BaP; and benz[a]anthracene, benzo[b]fluoranthene, chrysene and indeno[1,2,3-c,d]pyrene are all initiators of skin carcinogenesis in mice (U.S. EPA 1980c, IARC 1971, Habs et al. 1980).

Although BaP is a more active mutagen than any of the compounds in this group, mixed mutagenic responses have been reported for these compounds (Peter et al. 1979, Basler and Rohrborn 1978, Roszinky-Kocher et al. 1979, Simmon 1979, Simmon et al. 1979).[14]

Little information is available on other toxic effects associated with exposure to these compounds. One should not overlook possible augmentation of effects through synergistic or co-carcinogenic mechanisms, however, current understanding of the co-carcinogenesis process is not sufficient to allow estimation of potential human risk.

Exposure has not been well defined for any of these compounds. Benz[a]anthracene has been identified in cigarette smoke (Schmeltz et al. 1975) and this probably represents an important source of exposure. On the basis of the data of U.S. EPA (1980c) and White and Vanderslice (1980), inhalation exposures in urban areas are estimated to be generally in the range of 2-400 ng/day. Levels of benzo[g,h,i]perylene appear to be somewhat higher, although monitoring data are limited. The available information suggests that dietary exposure for those compounds is generally less than 50 ng/day.[15] On the basis of the data of Basu and Saxena (1978), drinking water exposures are generally less than about 5 ng/day, although no data are available for most of the compounds.

2.4.5 Aquatic Effects and Exposure

There are almost no data describing the acute toxic effects to aquatic organisms of the PAHs in this group. Benz[a]anthracene at a

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[14]These data are summarized in Volume IV of this report.
[15]This information is described in Volume IV of this report.

concentration of 1 µg/l caused 87% mortality in freshwater bluegill in 6 months (Brown et al. 1975). No other acute toxicity data for these PAHs were found.

A limited number of studies of sublethal and chronic effects have been found as described in Volume IV of this report. These are generally inadequate to define the concentration ranges associated with these effects.

Experiments conducted on a small number of species of aquatic invertebrates and lower vertebrates have shown that PAHs in the BaP group may cause tumor production and teratogenic and mutagenic effects (Neff 1979). Concentration levels associated with these effects were not given. Increased liver disease in benthic English sole has been shown to be associated with sediments contaminated with PAHs; however, no concentration data are available (Malins 1979).

The limited number of positive observations in STORET of the PAHs in this group indicates a concentration range in surface waters from 0.01 µg/l to 1500 µg/l; benzo[k]fluoranthene, with five observations ranging from 320 µg/l to 1500 µg/l, was consistently reported at higher levels than the other PAHs in this group (U.S. EPA 1980c). Other data sources generally report ambient water concentrations of these PAHs to be less than 1 µg/l (White and Vanderslice 1980). Sediment concentrations recorded in STORET are generally less than 1000 µg/kg; however, a few observations were reported above 1000 µg/kg. In the absence of quantitative data on toxicologic effects, it is not possible to assess the significance of exposure of aquatic biota to concentrations in this range.

The environmental conditions present in an aquatic system actually determine the extent of exposure to aquatic organisms. Though environmental models predict that most of the BaP (and probably the compounds in this group as well) in aquatic systems will be transported to the sediments, the bioavailability of sediment-bound PAHs is not well understood. Several studies have concluded that any PAHs that are actually taken up by fish come from the interstitial waters and from the water column (dissolved and/or adsorbed onto suspended solids); uptake from the sediment itself does not appear to be significant (Neff 1979).

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